1134-135 Plasma Renin Activity (PRA) and Acute Myocardial Infarction (AMI) – More Evidence for a Positive Association

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Background: High PRA is independently associated with increased risk of MI in hypertensives (NEJM 1991; 324: 1098). The present study further assesses the association of PRA to AMI in the acute setting.

Method: Upon entry to the emergency department, prior to any acute treatment, PRA was measured as part of the standard evaluation of 349 consecutive patients who were hospitalized for suspected MI.

Results: Diagnosis of AMI was confirmed/unconfirmed in 73/276. These groups did not differ in age (65.9 ± 0.2 vs 66.1 ± 0.9 yrs.), systolic (143 ± 4 vs 140 ± 2 mm Hg), or diastolic (81 ± 2 vs 81 ± 1 mm Hg) pressures. Mean PRA at entry was 3-fold higher in MI (1.2 vs 3.2 ng/ml/h; $p < 0.0001$). PRA, as a continuous variable, was found in multivariate analysis to be the predominant independent risk factor for AMI ($p < 0.00001$), followed by white race ($p < 0.02$), hypertension ($p = 0.02$), male gender ($p < 0.05$) and prior CABG ($p < 0.05$), controlling for other risk factors including hypercholesterolemia (NS) and prior drug therapy (NS). Moreover, in a separate multivariate analysis controlling for other risk factors, treatment with long-acting dihydropyridine calcium blockers was associated with a significant increase in AMI compared to treatment with β-blockers (OR = 3.2; CI 2.0 to 5.3).

Conclusion: PRA is considerably higher in AMI and is independently associated with increased risk of AMI. These findings give new support to the possibility that excessive activity of the renin system in this acute setting, as well as in ambulatory patients, plays a role in pathogenesis of MI in a substantial fraction of patients.

1134-136 Myocardial Infarction With and Without Coronary Artery Disease in African Americans

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We have reported previously from our laboratory regarding African American patients with chest pain and myocardial ischemia in the absence of epicardial coronary artery disease (CAD). Individuals with this syndrome are clinically indistinguishable from patients with CAD. We studied 153 consecutive patients (pts) with acute myocardial infarction seen at our hospital from Dec. 1996 to July 1997. 44 pts from this group underwent cardiac catheterization and 36 had CAD defined as greater than 50% luminal narrowing in at least one major epicardial vessel, 8 had normal coronary arteries (nl). There was no cocaine use in either group.

	men %	women %	trop* ng/dl	death %	DM* %	LVH %	EF
Nl	38	62	28.3	13	55	50	39.5
CAD	42	58	33.7	14	55	67	49.6

*normal coronaries (Nl), troponin I (trop), diabetes mellitus (DM)

There was no statistically significant difference in any of the above variables between individuals with angiographically documented CAD and normal coronary arteries. The degree of troponin elevation did not predict infarct size, severity of CAD, or mortality. These data suggest that myocardial ischemia and necrosis can occur in the absence of epicardial disease and this abnormality of the microvasculature is indistinguishable from "classic" CAD. Further prospective evaluation of the identification and optimal therapy of these patients is clearly warranted.

1135 Angiotensin Converting Enzyme Inhibitors and Receptor Blockers in Acute Myocardial Infarction

Tuesday, March 31, 1998, 3:00 p.m.–5:00 p.m.
Georgia World Congress Center, West Exhibit Hall Level
Presentation Hour: 4:00 p.m.–5:00 p.m.

1135-149 Aspirin Does Not Interact With ACE Inhibitors When Both Are Given Early After Acute Myocardial Infarction

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Background: Aspirin (ASA) and ACE inhibitors (ACEi) reduce mortality when given early after MI. Although there are pharmacological ways by which ASA could antagonize some of the beneficial effects of ACEi (e.g. inhibition of synthesis of vasodilating prostaglandins), evidences from large controlled trials are controversial.

Methods: We analyzed the data base of a recently performed overview on 98483 individual pt data allocated ACEi or no-ACEi within 36 h from the onset of symptoms of MI in 4 trials, CCS-1, CONSENSUS II, GISSI-3 and ISIS-4.

Results: Out of 96712 pts analyzable, 10228 (10.6%) did not receive ASA at entry. Overall ACEi reduced 30-day mortality from 7.6% to 7.1% ($p = 0.004$); mortality in pts receiving ASA was reduced by ACEi from 6.7% to 6.3% (proportional % reduction ±SD: 6.3 ± 2.7), and in pts not receiving ASA from 15.1% to 13.8% (proportional % reduction ±SD: 10.0 ± 5.3). Mortality reduction in the 2 groups was not significantly different (χ^2 for heterogeneity = 0.4). The existence of an interaction between ASA and ACEi on 30-day mortality was also tested by multivariate analysis adjusted for confounding variables at entry (age, Killip class, heart rate): the interaction was not statistically significant ($\chi^2 = 0.01$). The risk of persistent hypotension was increased by ACEi from 9.4% to 17.9% in pts with ASA and from 10.0% to 18.3% in pts without ASA. The risk of renal dysfunction was increased from 0.6% to 1.3% in pts with ASA and from 0.7% to 1.4% in pts without ASA. No interaction between ASA and ACEi was found for these two adverse events both on univariate and multivariate analysis.

Conclusions: ASA neither attenuates the mortality benefit of early ACEi after MI, nor increases the risk of major adverse events. Therefore, ACEi can be safely given early after MI even when ASA is being given.

1135-150 Effects of Angiotensin Converting Enzyme Inhibition on Cardiopulmonary Baroreflex Sensitivity in Patients With Acute Myocardial Infarction

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Background: Cardiopulmonary reflexes (CP) tonically inhibit sympathetic outflow and are blunted in patients with congestive heart failure. However, no data are available about the effects of angiotensin converting enzyme (ACE) inhibition on CP in patients with acute myocardial infarction (MI). The aim of the study was to evaluate the effects of ACE inhibitor (quinapril) on CP in patients with uncomplicated MI.

Methods: Fifteen patients with uncomplicated MI (group A) underwent CP sensitivity evaluation 5 days after the onset of MI and after 10 days of quinapril therapy. Fifteen additional MI patients (group B: control group) were evaluated at the same time interval before and after placebo administration to identify spontaneous CP variation. CP was assessed by the response of forearm vascular resistance induced by lowering central venous pressure through lower body negative pressure to-10 mmHg.

Results: Before quinapril administration, there were no significant differences in hemodynamic variables, ejection fraction (59 ± 6 vs 60 ± 11%), CP (20 ± 14 vs 14 ± 5%), plasma noradrenaline (479 ± 285 vs 354 ± 111 pg/ml) and plasma renin activity (1.2 ± 1.1 vs 2.0 ± 1.4 ng/ml/hr) between group A and group B. After quinapril administration, CP in group A was significantly higher than that in group B (47 ± 24 vs 24 ± 9%, $p < 0.05$). Moreover, the amount of increase in CP was larger in group A than that in group B (26 ± 21 vs 10 ± 5, $p < 0.05$). In group A, plasma noradrenaline decreased (479 → 392 pg/ml) and plasma renin activity increased (1.2 → 5.7 ng/ml/hr), but not in groupB.

Conclusions: Quinapril improves CP in patients with uncomplicated MI. This improvement was associated with a reduction in sympathetic outflow and may contribute to the beneficial effects of ACE inhibitors in patients with acute MI.

1135-151 Effects of Early ACE-Inhibition in Patients With AMI and Arterial Hypertension

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ACE inhibitors (ACEI) have been demonstrated to be largely effective for the treatment of both hypertension and coronary artery disease. The present study was carried out as post-hoc analysis of the SMILE data base to evaluate the influence of history of arterial hypertension on the efficacy of 6-week treatment with zofenopril given early (<24 hours) to patients with acute anterior myocardial infarction (AMI). The main outcome measures were: 6-week combined occurrence of death (D) and severe refractory CHF (S-CHF) as well as 1-year mortality (1Y-D). Normotensive (NBP, n.876) and hypertensive (HBP, n.566) patients were largely comparable at baseline. In the HBP group the 6-week prevalence of death (8.2 vs 5.8%), S-CHF (5.8 vs 2.3%) and death + S-CHF (14.1 vs 7.9%) was increased when compared with the NBP group (2p = 0.001) as was 1-year mortality (19.6% vs 16.2%; 2p = 0.05).